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RESEARCH ARTICLE

An agent-based model for integrated emotion regulation and contagion in socially affected decision making



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KEYWORDS

Emotion regulation;
Emotion contagion;
Decision making;
Computational social agent
model

Abstract

This paper addresses an agent-based computational social agent model for the integration of emotion regulation, emotion contagion and decision making in a social context. The model integrates emotion-related valuing, in order to analyse the role of emotions in socially affected decision making. The agent-based model is illustrated for the interaction between two persons. Simulation experiments for different kinds of scenarios help to understand how decisions can be affected by regulating the emotions involved, and how these emotions are affected by emotion regulation and contagion.

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Introduction

Traditionally emotions have often been considered as standing in the way of adequate decision making. In recent developments on human decision making from a cognitive and neurological perspective a more constructive role of emotions has been found. For the case of individual decision making in particular, this concerns the role of emotions in a process in which a number of action options are considered, for each of which effects are predicted by internal

simulation. By valuing these predicted effects a choice is made (e.g., Phelps, Lempert, & Sokol-Hessner, 2014; Treur & Umair, 2011). In this valuation process emotions play a crucial role: those action options for which the predicted effects associate to a more positive feeling will be valued higher, and therefore will be chosen more often.

In a social context, often decision making processes of different individuals affect each other, by social contagion processes (e.g., Bosse, Duell, Memon, Treur, & van der Wal, 2014; Bosse et al., 2012). A specific form of social contagion relevant in such socially affected decision making processes is emotion contagion. By expressing their emotions associated to different decision options, individuals

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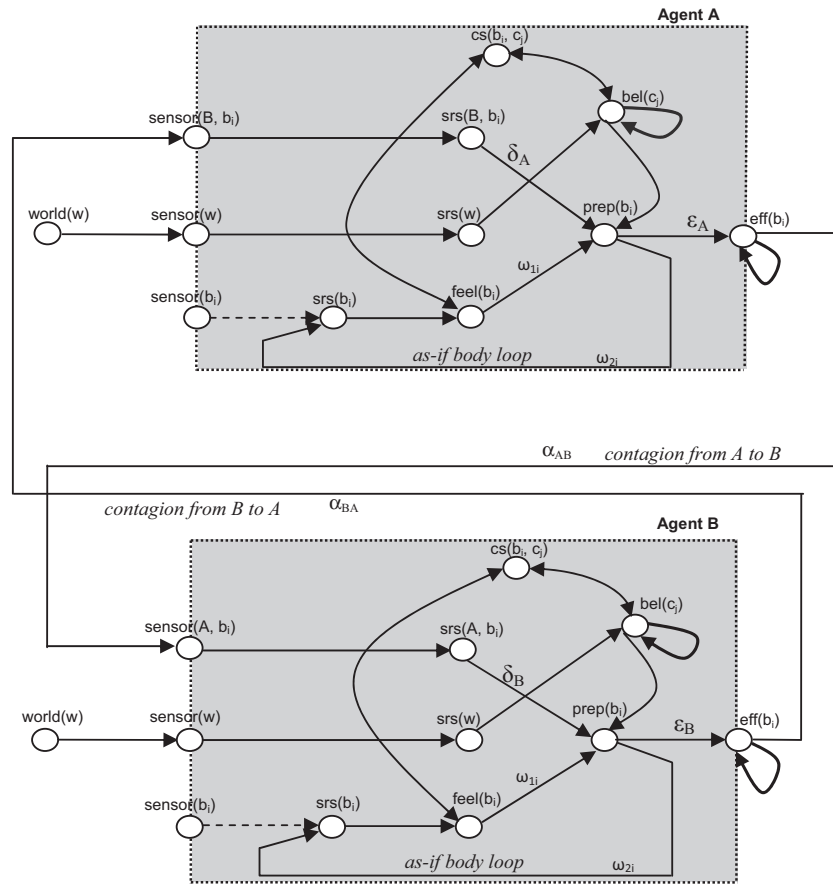


Fig. 1 Overview of the computational model.

affect other individuals in their emotions for these options. Through this an instantaneous social effect on the choice of an action occurs.

In this social decision making process also a form of learning takes place. When a considered action co-occurs with a positive emotion, the association between this option and the emotion will be strengthened, so that in future situations the initial emotional effect is stronger. In this way such social contagion processes may also affect decisions for individuals made in the future.

The strength of how emotions in an individual develop and are transferred to other individuals (contagion), also depends on the extent to which emotions are regulated. Some persons are expressing their emotions less than others. For example, when a person applies a very strong form of emotion regulation so that only a neutral face and body are shown, emotion contagion will not take place, and therefore decision making of others is not affected by such an emotion. Also, for an individual observing the emotion of another individual, if this received emotion is strongly regulated, this may reduce the social effect on the decision making. Such strong emotion regulation may make it more difficult to reach joint decisions in a natural manner.

In this paper a social agent model is presented that covers how socially affected decision making relates to emotion

contagion in interaction with emotion regulation. The work presented here is an extension of [Manzoor and Treur \(2013\)](#). First, in the second section the model itself is explained in some detail. In the third section it is illustrated by means of an example simulation showing how the model works. Next, in the fourth section more refined explorations are discussed of different scenarios showing the role of emotion regulation in the decision making. Finally, fifth section is a discussion.

A computational social agent model integrating emotion regulation and contagion

As discussed above, in a social context decisions of an individual are not often made independent of other individuals, due to the role of social contagion, in particular of emotions related to decision options. Moreover, these emotions usually are also subject to internal regulation processes. To explore the combination of such processes, the social agent model for socially decision making (see [Fig. 1](#) for an overview) presented here is based on the three key principles, namely:

- emotion-related valuing of decision options,
- emotion contagion,
- emotion regulation.

Table 1 Overview of the impacts on the states.

State	Notation	Impacts on this state	Combined impact values
World state for w	$world(w)$	—	$world(w) \cdot \omega(world(w), sensor(w))$
Sensor state for w	$sensor(w)$	$world(w)$	$sensor(w) \cdot \omega(world(w), sensor(w))$
Sensory representation for w	$srs(w)$	$sensor(w)$	$srs(w) \cdot \omega(sensor(w), bel(c_k)) + bel(c_k) \cdot \omega(bel(c_j), bel(c_k)) + cs(b_i, c_j) \cdot \omega(cs(b_i, c_j), bel(c_j))$
Belief state for c_j	$bel(c_j)$	$srs(w), bel(c_k), cs(b_i, c_j)$	$bel(c_j) \cdot \omega(bel(c_j), prep(b_i)) + feel(b_i) \cdot \omega(feel(c_i), prep(b_i)) + srs(b_i) \cdot \omega(srs(b_i), prep(b_i))$
Preparation state for b_i	$prep(b_i)$	$bel(c_j), feel(b_i), srs(b_i)$	$prep(b_i) \cdot \omega(prep(b_i), srs(b_i))$
Sensory representation state for b_i	$srs(b_i)$	$prep(b_i)$	$srs(b_i) \cdot \omega(srs(b_i), feel(b_i)) + cs(b_i, c_j) \cdot \omega(cs(b_i, c_j), feel(b_i)) + cs(b_i, c_k) \cdot \omega(cs(b_i, c_k), feel(b_i))$
Feeling state for b_i	$feel(b_i)$	$srs(b_i), cs(b_i, c_j), cs(b_i, c_k)$	$prep(b_i) \cdot \omega(prep(b_i), eff(b_k)) + eff(b_k) \cdot \omega(eff(b_k), eff(b_i))$
Effector state for b_i	$eff(b_i)$	$prep(b_i), eff(b_k)$	$eff(b_i) \cdot \omega(eff(b_i), sensor(b_i))$
Sensor state for another agent and b_i	$sensor(b_i)$	$eff(b_i)$	$sensor(b_i) \cdot \omega(sensor(b_i), srs(b_i))$
Sensory representation state for another agent and b_i	$srs(b_i)$	$sensor(b_i)$	$feel(b_i) \cdot \omega(feel(b_i), cs(b_i, c_j)) + bel(c_j) \cdot \omega(bel(c_j), cs(b_i, c_j))$
Control state for b_i and c_j	$cs(b_i, c_j)$	$feel(b_i), bel(c_j)$	

The basic model of decision making based on emotion-related valuing is adopted from the model described in [Treur and Umai \(2011\)](#), also see [Phelps et al. \(2014\)](#), [Rolls \(2013\)](#), p. 704, and [Rolls \(2014\)](#). This is an approach to decision making in which for each option it is determined to which extent its predicted effect associates to a positive feeling. Also the Hebbian learning mechanism is adopted from this model: some of the connections in the model become stronger when the connected states are activated at the same time. The model for emotion contagion is adopted from [Bosse, Duell, Memon, Treur, and Van Der Wal \(2009\)](#) and [Bosse et al. \(2012\)](#).

The model for emotion regulation is based on recent neurological literature which addresses how emotion regulation takes place by an interaction between prefrontal cortex and amygdala ([Gross, 2015a, 2015b](#); [Kim et al., 2011](#); [Ochsner & Gross, 2014](#); [Phelps, Delgado, Nearing, & LeDoux, 2004](#); [Sotres-Bayon, Bush, & LeDoux, 2004](#); [Yoo, Gujar, Hu, Jolesz, & Walker, 2007](#)).

Several findings indicate that less adequate emotion regulation correlates to lower activity in prefrontal cortex areas and less strong connections from amygdala to prefrontal cortex ([Kim et al., 2011](#)). Moreover, strong indications have been found that REM-sleep strengthens both activation of prefrontal cortex and emotion regulation ([Gujar, McDonald, Nishida, & Walker, 2011](#)).

The dashed arrow from $sensor(b_i)$ to $srs(b_i)$ is symbolic and does not do anything in the scenarios as described. [Table 1](#) shows which impacts contribute to the values of the different states at any time point t , as can also be observed from [Fig. 1](#).

Although the model is more general, for the sake of simplicity, in this paper only two agents are considered. Agents are described in terms of the dynamics of their internal states, indicated by circles in the dotted boxes, and their interaction states, indicated by circles on the dotted line (see [Fig. 1](#)). The sections below elaborate the role of the various internal states of the model.

World, sensor, and sensory representation states

An agent observes the world state $world(w)$ through the sensor state $sensor(w)$. This world state represents the current situation in which the agent may be facing, for example, boredom, fatigue, or a need to adapt its life style. The very first step in the process is to generate the internal sensory representations of the world state. It depends on the agent that the sensory representation $srs(w)$ is associated to different belief states $bel(c_j)$ according to different connection strengths, as some beliefs hold to be true for certain people and for others they might not.

Beliefs, feelings, preparations, and effectors

Belief states $bel(c_j)$ are considered as alternative interpretations of observations from the world. Therefore they suppress each other through mutual inhibition. Moreover, they affect the decision making process of an agent through different connection strengths to the preparation states $prep(b_i)$ for different action options b_i . The internal preparation for a certain action option does not only depend on the

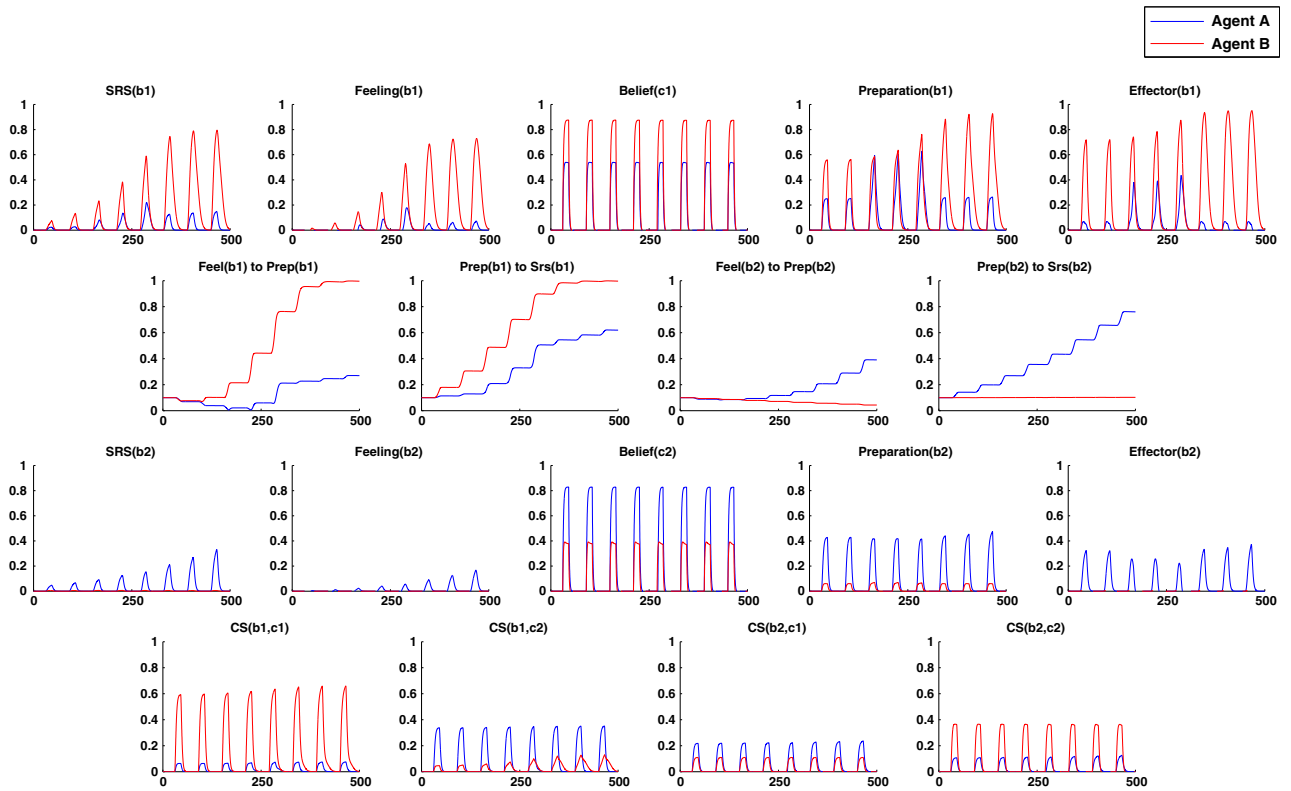


Fig. 2 Effects of contagion with regulation on behaviour and learning of behaviour. The regulation has an effect on the learning of behaviour but the three blue spikes in the middle of the graphs for preparation(b_1) and effector(b_1) show some effects of contagion from agent B to agent A, but these spikes disappear as soon as the social interaction ends (last three iterations) which shows that the learning was not effective, and the social influence was only temporary for the duration of the social interaction.

beliefs but also on the feelings associated with the option. Before performing an action, a (positive) feeling state $feel(b_i)$ for the option b_i is affected by a predictive as-if body loop (Damasio, 1994) via the sensory representation state $srs(b_i)$. During processing the activation level of $feel(b_i)$ indicates the strength of the feeling. It is this strength (if not low) that can strengthen the preparation: the higher the strength the more influence on the preparation. This gives a sense of valuing a prediction about the option before executing an action to perform it; this has some similarity to utility-based decision making, where a utility corresponds to the extent in which the predicted effect leads to a positive feeling. In a scenario where emotion regulation is present, the activation level of the feeling state $feel(b_i)$ also depends on the control state $cs(b_i, c_j)$.

The feeling state $feel(b_i)$ also affects the preparation state $prep(b_i)$, which makes the as-if body loop recursive. The way in which an agent's decision to execute a certain action in the outside world is affected by the (recursive) as-if body loop which portrays the effects of the associated feeling on the preparation of the action option. There may or may not be a one to one correspondence between the beliefs and the feelings. It may be the case that the as-if body loop makes an adjustment of the action option indicated by the beliefs. But in more coherent cases, for example, a strong belief about a decision option b_i may go together with a strong positive feeling attached to that

option and for a weak belief the other way around. It is possible for an agent to have mixed feelings about the different options, but still when it comes to select any one of the two or more mutually exclusive options, by a form of mutual inhibition (by negative mutual connection weights) the effector state $eff(b_i)$ will become significant for only one option. This describes how an agent's feeling and belief play an important role in a decision making process. If the agent is operating in a social environment then the role of contagion has to be taken into account as well because it could alter the feelings of an agent.

Contagion: channel strength, expressiveness, and openness

Within the collective decision making model an additional mechanism for contagion has been incorporated, based on mirroring of the preparation states (also see Bosse et al., 2012, 2009). An important element is the contagion strength γ_{BA} from person B to person A . This indicates the strength by which a preparation state S (for an option b_i) of A is affected by the corresponding preparation state S of B . It depends on characteristics of the two persons: how expressive B is, how open A is, and how strong the connection channel from B to A is. In the model it is defined by

$$\gamma_{BA} = \varepsilon_B \alpha_{BA} \delta_A \quad (1)$$

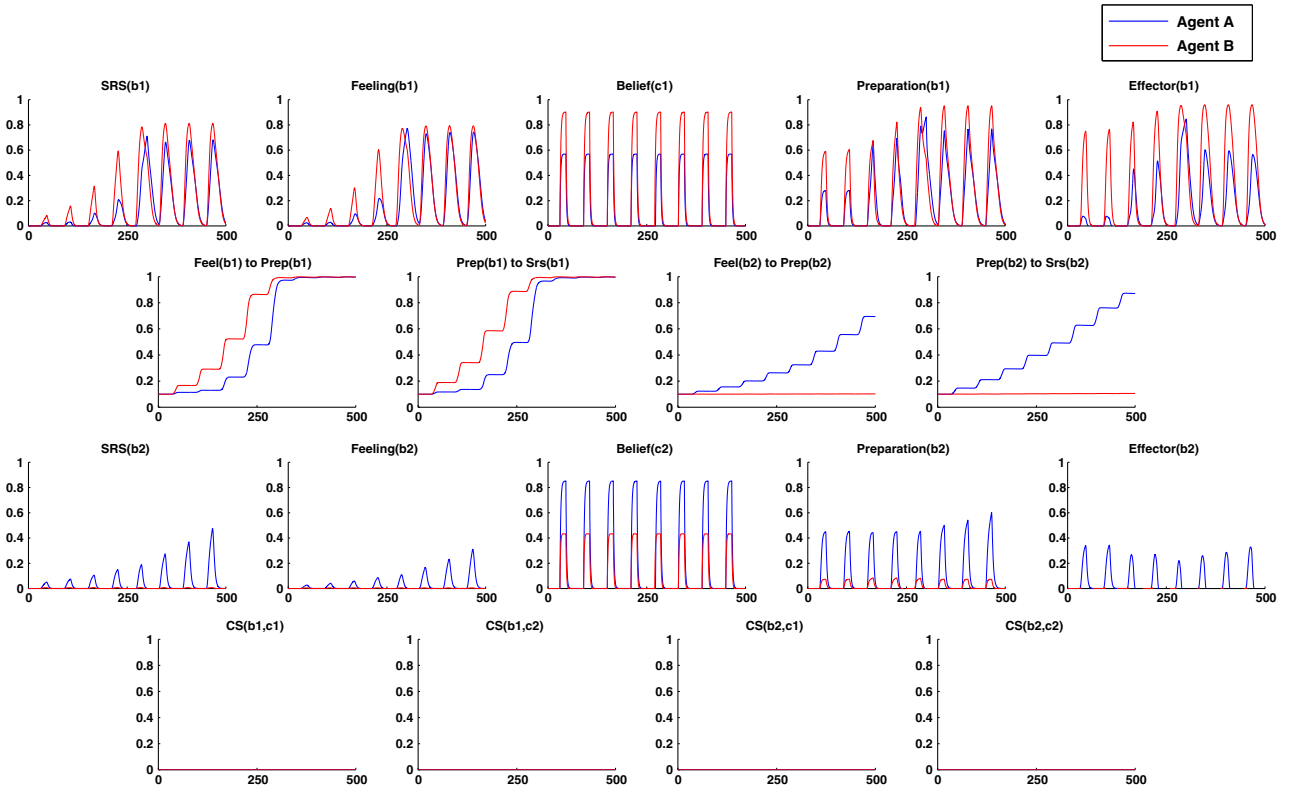


Fig. 3 Effects of contagion without regulation on behaviour and learning of behaviour. In the absence of regulation the graphs show a gradual increase (convergence) of blue spikes (agent A) which is the result of strong contagion from agent A to agent B. In this case in the third phase the behaviour of agent A still persists, which shows that in the social context (in the second phase) new behaviour has been learnt that is also displayed outside the social context. The following connection weights are 0: $wgt_bel_i_cs$, $wgt_feel_i_cs$, $wgt_cs_feel_i$.

Here, ε_B is the *expressiveness* of B , δ_A the *openness* of A , and α_{BA} the *channel strength* from B to A . Note the labels in Fig. 1 for these concepts. The level q_{SA} of preparation state S in agent A (with values in the interval $[0, 1]$) over time is determined as follows. The overall contagion strength γ_A from the rest of the group towards agent A is $\gamma_A = \sum_{B \neq A} \gamma_{BA}$. The aggregated impact q_{SA}^* of all these agents upon state S of agent A is the following weighted average:

$$q_{SA}^*(t) = \sum_{B \neq A} \gamma_{BA} q_{SB}(t) / \gamma_A \quad (2)$$

This is an additional external impact on the preparation state S of A , which has to be combined with the impact from the internal emotion-related valuing process. Note that for the case that there is only one other agent, this expression for $q_{SA}^*(t)$ can be simplified to $q_{SB}(t)$.

Emotion regulation: control, beliefs, feelings

Over the years several strategies have been proposed in the literature regarding emotion regulation. Broadly speaking, they are categorised into two major types: the ones that can be used before an emotion response has an effect on the behaviour (antecedent-focused strategies) and the others in situations where the emotion response already

comes into effect (response-focused strategies) (Gross, 1998, 2015a, 2015b; Ochsner & Gross, 2014). In the current paper the focus is on antecedent focused strategies, and in particular on reappraisal. As discussed earlier, a higher activation level of a preparation state $prep(b_i)$ for a certain option b_i depends on the beliefs $bel(c_j)$ and the feeling $feel(b_i)$. Thus, a strong belief and a positive feeling together support a choice for an option b_i (see also Section 'Beliefs, feelings, preparations, and effectors').

Since in this paper the regulation is based on antecedent focused strategies, and more in particular reappraisal, the emotion regulation is modelled as a dynamic interaction between the following three states of the model: $cs(b_i, c_j)$, $bel(c_j)$, $feel(b_i)$. The emotion regulation mechanism uses negative weights from the control state $cs(b_i, c_j)$ to the belief state $bel(c_j)$ and the feeling state $feel(b_i)$. Depending on the characteristics of a person the emotion regulation mechanism works strong or less strong (represented by higher or lower values for these negative weights). In simulation scenarios this has been varied for both agents.

Hebbian learning

In the model the connection strengths of two types of connections are adapted by Hebbian learning (Hebb, 2002): from preparation state $prep(b_i)$ to sensory representation state

Table 2 Different types of effects of contagion.

	No regulation	Regulation
No learning	Direct effects of contagion without regulation on behaviour (Section 'Direct effects of contagion without regulation on behaviour, and without learning')	Direct effects of contagion combined with regulation on behaviour (Section 'Direct effects of combined contagion and regulation on behaviour, without learning')
Learning	Effects of contagion without regulation on learning of behaviour (Section 'Effects of contagion without regulation on behaviour and learning of behaviour')	Effects of contagion combined with regulation on learning of behaviour (Section 'Effects of contagion combined with regulation on behaviour and learning of behaviour')

$srs(b_i)$, and from feeling state $feel(b_i)$ to preparation state $prep(b_i)$. From a Hebbian perspective, strengthening of a connection over time may take place when both nodes are often active simultaneously ('neurons that fire together wire together'). The principle goes back to [Hebb \(2002\)](#), and has got some attention over time, also computationally, but has recently gained even more interest by more extensive empirical support and more advanced mathematical formulations and applications (e.g., [Gerstner & Kistler, 2002](#)). As Hebbian learning depends on the activation levels of the connected states, a positive evaluation of a performed action has a positive effect on the learning, as in this case the sensory representation state $srs(b_i)$ gets a higher activation level. When by the Hebbian learning mechanism the connection strength from the connection within the as-if body loop $prep(b_i)$ to $srs(b_i)$ has increased, this implies that the association of the option to the predicted feeling will become stronger, so for a next occasion when the item is encountered the valuing of the option before a decision is made will be higher. In addition, Hebbian learning also enables to increase the impact a given feeling level has for a certain option: the connection strength from $feel(b_i)$ to $prep(b_i)$. These are two ways in which the role of valuing of options are adapted over time. Note that this is a different way of learning, for example, compared to strengthening connections from sensory representation of a stimulus to preparation, as would be the case in stimulus–response learning.

For the connections from $prep(b_i)$ to $srs(b_i)$ and from $feel(b_i)$ to $prep(b_i)$ their strengths are adapted using the following *Hebbian learning rule*, taking into account a maximal connection strength 1, a *learning rate* η , and an *extinction rate* ζ (usually taken small):

$$\begin{aligned} \omega((prep(b_i), srs(b_i)))(t + \Delta t) = & \omega((prep(b_i), srs(b_i)))(t) + [\eta * prep(b_i) \\ & \times (t) * srs(b_i) * (1 - \omega((prep(b_i), srs(b_i)))(t)) \\ & - \zeta * \omega((prep(b_i), srs(b_i)))(t)] \Delta t \end{aligned} \quad (3)$$

$$\begin{aligned} \omega((feel(b_i), prep(b_i)))(t + \Delta t) = & \omega((feel(b_i), prep(b_i)))(t) \\ & + [\eta * feel(b_i)(t) * prep(b_i) \\ & * (1 - \omega((feel(b_i), prep(b_i)))(t)) \\ & - \zeta * \omega((feel(b_i), prep(b_i)))(t)] \Delta t \end{aligned} \quad (4)$$

A similar Hebbian learning rule can be found in [Gerstner and Kistler \(2002, p. 406\)](#). By the factor $1 - \omega((prep(b_i), srs(b_i)))(t)$ respectively $1 - \omega((feel(b_i), prep(b_i)))(t)$ the learning

rule keeps the connection strengths bounded by 1 (which could be replaced by any other positive number). When the extinction rate is relatively low, the upward changes during learning are proportional to the activation levels of both connected states and maximal learning takes place when both are 1. Whenever one of these activation levels is 0 (or close to 0) extinction takes over, and the connection strength slowly decreases.

Dynamics of states

The dynamical modelling approach was inspired by [Beer \(1995\)](#). The activation level of a state is determined by the impact of all the incoming connections from other states thereby being multiplied by their corresponding connection weights. In particular, for a state causally affected by multiple other states, to obtain their combined impact, first the activation levels V_i for these incoming state are weighted by the respective connection strengths ω_i thus obtaining $X_i = \omega_i V_i$, and then these values X_i are combined, using a combination function $f(X_1, \dots, X_n)$. In the context of current paper the combination function is based on the following logistic threshold function:

$$f(X_1, \dots, X_n) = th(\sigma, \tau, X_1 + \dots + X_n) \quad (5)$$

with

$$th(\sigma, \tau, X) = \left(\frac{1}{1 + e^{-\sigma(X - \tau)}} - \frac{1}{1 + e^{\sigma\tau}} \right) (1 + e^{-\sigma\tau}) \quad (6)$$

An alternative formal specification of the model in terms of the hybrid LEADSTO format ([Bosse, Jonker, Van Der Meij, & Treur, 2007](#)) is shown in the Appendix A.

Illustration of the model by an example simulation experiment

This section illustrates the model by an example scenario. The graphs in [Fig. 2](#) give an idea of how the model behaves when all the elements discussed above are working together to achieve a fully integrated process (integrating the contagion, regulation and decision making). As discussed in more detail in Section 'Further analysis of the model by simulation experiments', to have an idea the scenario can be assumed to involve two action options indicated by b_1 and

Table 3 Connection strengths used in the simulation scenarios.

From	To											
	world(w)	ss(w)	srs(w)	bel(c _j)	prep(b _j)	eff(b _i)	ss(b _i)	srs(b _i)	feel(b _i)	ss(X, b _i)	srs(X, b _i)	cs(b _i , c _j)
world(w)		1										
ss(w)			1, 1									
srs(w)			1, 1									
bel(c _j)				0.5, 0.8 0.9, 0.4 −0.1, −0.1 −0.1, −0.1	0.9, 0.8 0.9, 0.5							0.1, 0.5 0.1, 0.5 0.5, 0.1 0.5, 0.1
prep(b _j)						0.8, 1		0.7, 0.9 0.9, 0.7				
eff(b _i)						−0.4, −0.4 −0.4, −0.4				0.4, 0.9		
ss(b _i)												
srs(b _i)									1, 0.6 0.9, 0.6			
feel(b _i)					0.7, 0.9 0.9, 0.7							0.1, 0.1 0.1, 0.1 0.1, 0.1 0.1, 0.1
ss(X, b _i)											1, 1	
srs(X, b _i)					0.9, 0.5							
cs(b _i , c _j)				−1, −1 −1, −1 −1, −1 −1, −1					−0.1, −0.1 −0.1, −0.1 −0.1, −0.1 −0.1, −0.1			

Table 4 Parameter values used in the simulation scenario.

	Prep	srs(b)	feel	bel1	cs(b _i , c _j)	effector	ss(X, b _i)	srs(X, b _i)
τ	4	3	4	4	4	6	3	3
σ	0.7	0.3	0.3	0.2	0.2	0.4	0.4	0.4

b_2 : option 1 (b_1), to go for exercising at a sport school or option 2 (b_2), watch TV. Agent A tends to go for option 2 first, but due to contagion from agent B finally decides to go for option 1. In the graphs, states of agent A are depicted in blue colour and agent B in a red colour. The y-axis shows the activation level or connection strength of a state or connection, in the interval of $[0, 1]$ and the x-axis represents time (in minutes). An activation level determines the intensity of a state at a particular time point, 1 being the highest activation level and 0 the lowest. In the scenarios an oscillatory stimulus has been chosen. This is because for learning it is assumed that there are different instances of such processes recurring over time; at different points in time the agent encounters such a situation and performs some action and also learns from the situation. Moreover, three phases are considered in the scenario. First a phase in which the agents have no social interaction (two iterations), then a phase in which social interaction takes place (next three iterations), and finally, in the last three iterations again no social interaction takes place. In this last phase it can be seen whether in the social context in the second phase something has been learnt that persists over time.

In this case b_1 represents a good option. The first row in Fig. 2 illustrates two types of plots the second row represents the strengths of connections (Hebbian learning) and the rest are activation levels of different states (this also applies to the other figures with simulation results). For example, the maximum and minimum values of the learning connection $feel(b_1)$ to $prep(b_1)$ (subplot row 2 position 1) are 0.2704 and 0.058 at time point 475, and 167 respectively for agent A. The simulation shows the behaviour cycle for eight days and each iteration is performed for 15 min. The regulation mechanism in Fig. 2 is active for both agents, and it can be seen that emotion regulation in agent B suppresses the expressiveness that affects the contagion mechanism which in turn prevents the learning process for the good behaviour (b_1) to take place over a period of time in agent A (second row in Fig. 2). Emotion regulation works in both agents, which is evident in Fig. 2, showing that agent A's learning for option b_2 is also affected. The bottom row in Fig. 2 shows the activation levels of control state $cs(b_i, c_j)$; the four graphs represent the control states for the different combinations of feeling (b_i) and belief (c_j). The parameter values are given in Tables 3 and 4, apart from the following values for beliefs to control states and vice versa for agent A. These are $wgt_bel_cs = [0.1, 0.3; 0.3, 0.1]$ and $wgt_cs_bel = [-0.1, -0.1; -0.1, -0.1]$. For agent B, $wgt_bel_cs = [0.5, 0.1; 0.1, 0.7]$ and $wgt_cs_bel = [-0.1, -0.1; -0.1, -0.1]$. The notation wgt_bel_cs means connection weight between bel_i and cs . The reason for these choices is to be able to illustrate a fully integrated process with modest emotion regulation.

Furthermore, Fig. 3 shows results for a scenario without regulation. Here a comparison can be made with Fig. 2 to

examine how emotion regulation is able to affect the learning process in the context of a social interaction.

Fig. 3 shows that the contagion mechanism helps agent A to gradually adapt to good behaviour (b_1) and even after the contagion is stopped the execution of good behaviour is stronger compared to not so good (b_2). Note that the contagion is two way (from B to A and vice versa) as it would be in a real life situation, but in this paper the scenarios have been chosen in such a manner that agent B has more influence on agent A than the other way around (the channel strength α_{BA} from agent B to agent A is chosen higher than α_{AB} from agent A to agent B). More detailed discussion about the simulation results shown here is given in Section 'Further analysis of the model by simulation experiments'.

Further analysis of the model by simulation experiments

To analyse the different aspects of the model, a number of more focused experiments have been conducted. As the model combines effects of contagion and regulation, in particular it is of interest to explore in how far the combined effects differ from effects in cases in which only contagion takes place and no regulation. Moreover, relevant effects can be distinguished according to direct effects on behaviour itself and effects on learning of behaviour. Therefore in this section, four simulation experiments are discussed, according to the scheme depicted in Table 2.

In order to understand the scenario better the following real life context is considered, a context in which the model could be applicable. All simulations are based on the following scenario, but with some variations. For example it is possible that a person does not have contact with another person; in that case contagion does not occur. Since learning is involved besides regulation and contagion, it is assumed that there are different phases of the processes which occur periodically during a course of eight days for 15 min: two days without social interaction, followed by three days with social interaction, followed by three days without social interaction. Each day the agent encounters the situation, performs some action and also learns.

People often feel tired when they return home after work. Different kinds of activities are possible to relax body and mind so that one can prepare for the next day's routine! In the current scenario only two options are considered (to avoid a complex scenario, although more than two options are possible in a real life situation) to be available to the persons. The options are: choice 1, to relax body and mind with some physical activity going for exercise at the nearest sport school, or choice 2, stay at home and watch TV. The scenario assumes that two persons interact with each other through some kind of social media. Person A is friend of person B who is sitting

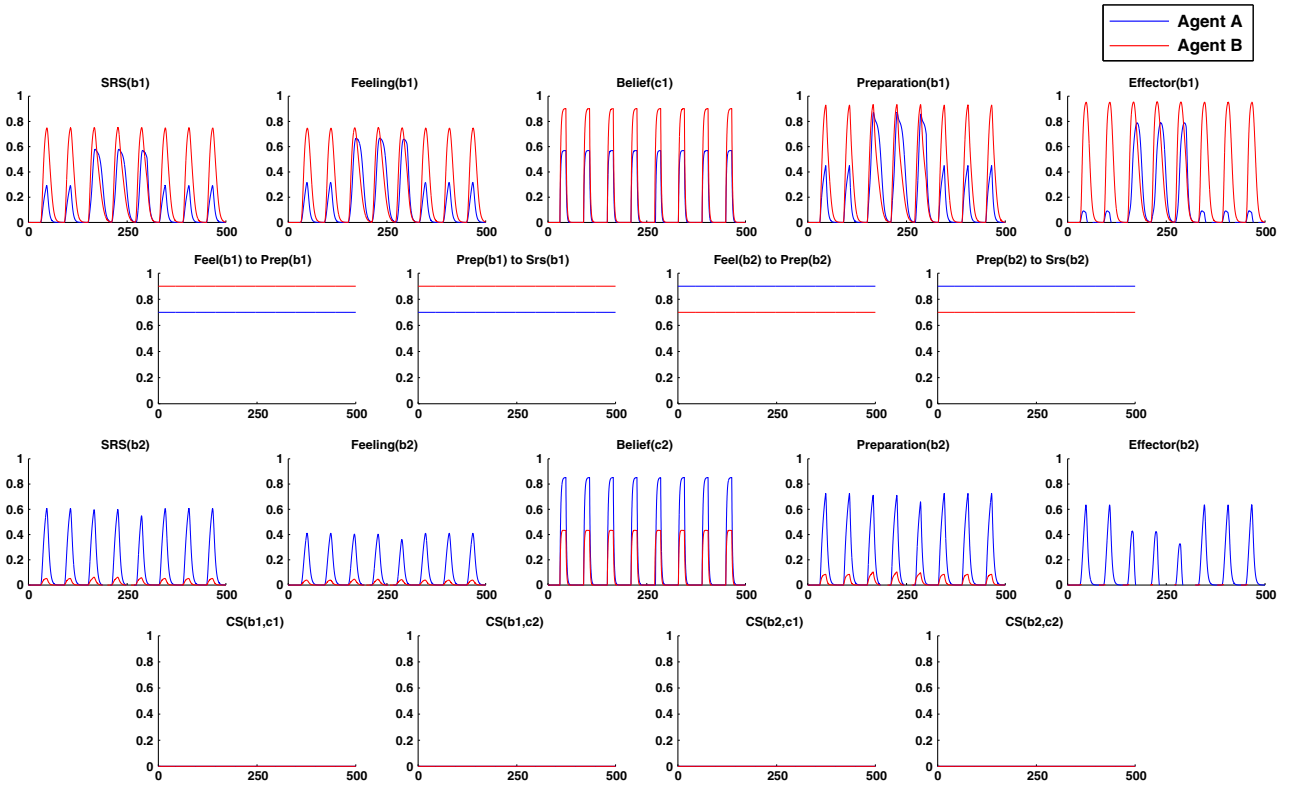


Fig. 4 Effects of contagion on behaviour without regulation and without learning. This simulation is performed by making the following connection weights to 0: wgt_bel_cs , wgt_cs_bel , wgt_feel_cs , wgt_cs_feel . Since no learning occurs in this case the values for the learning connections $feel(b_i)$ to $prep(b_i)$ and $prep(b_i)$ to $srs(b_i)$ are set at constant values 0.7 and 0.9 for agent A and for agent B at 0.9 and 0.7 respectively. The three blue peaks in the centre of the plot of $effector(b_1)$ shows the effects of contagion on choices for option 1.

on the couch after a hectic working day; it seems watching TV is a good option for her and initially she decides to stay at home but after being affected by her friend she chooses for the sport school.

Two agents play their part in the scenario, it is assumed that agent B plays the role of a good friend who persuades agent A to opt for the good option. A good option could be to visit a sport school, going for jogging, or opting for active transport, e.g., biking or walk. For all figures in this section the y-axis corresponds to the activation levels of a state or connection strengths in case of learning connections ($feel(b_i)$ to $prep(b_i)$ and $prep(b_i)$ to $srs(b_i)$) and the x-axis represents time in minutes. As before, in the graphs, states of agent A are depicted in a blue colour and the other one (agent B) in a red colour. Settings for the parameter values and for the weights are given in Table 3. The values were chosen so that qualitatively the generated patterns coincide with patterns described in (qualitative) literature; choosing these values was done by manual parameter tuning. Values for threshold and steepness parameters are specified in Table 4. Note that by assigning zero weights to connections from $cs(b_i, c_j)$ to $bel(c_j)$ and $feel(b_i)$ it is realised that no regulation takes place. The initial values of the states are set to 0, learning connections (from $prep(b_i)$ to $srs(b_i)$ and from $feel(b_i)$ to $prep(b_i)$) usually start at weight 0.1 but in cases

when no learning takes place the connections have been given (higher) fixed values (see Table 3). In Table 3 the first item of each cell represents the values belonging to agent A and the second item to agent B. If the regulation is working, appropriate values are set for the connections from $cs(b_i, c_j)$ to $bel(c_j)$, $feel(b_i)$ and vice versa; alternatively they are all set to zero. For the cases in which learning of behaviour is involved, the weights of the connections from $feel(b_i)$ to $prep(b_i)$ and from $prep(b_i)$ to $srs(b_i)$ are initially assigned the value 0.1. The value for update speed parameter for all states is 0.5.

Direct effects of contagion without regulation on behaviour, and without learning

As Fig. 1 shows, the world state $world(w)$ triggers preparations for some action options in each agent. Initially for agent A option 2 dominates, and for the second agent B it is the other way around. These tendencies relate to the specific connection settings between the sensory representations, beliefs and the preparation states, as can be seen in Table 3. The agents have also feelings associated with both choices, based on similar kinds of weight values given in Table 3. The results for this case are illustrated in Fig. 4. For example, agent B has a strong (positive) emotional asso-

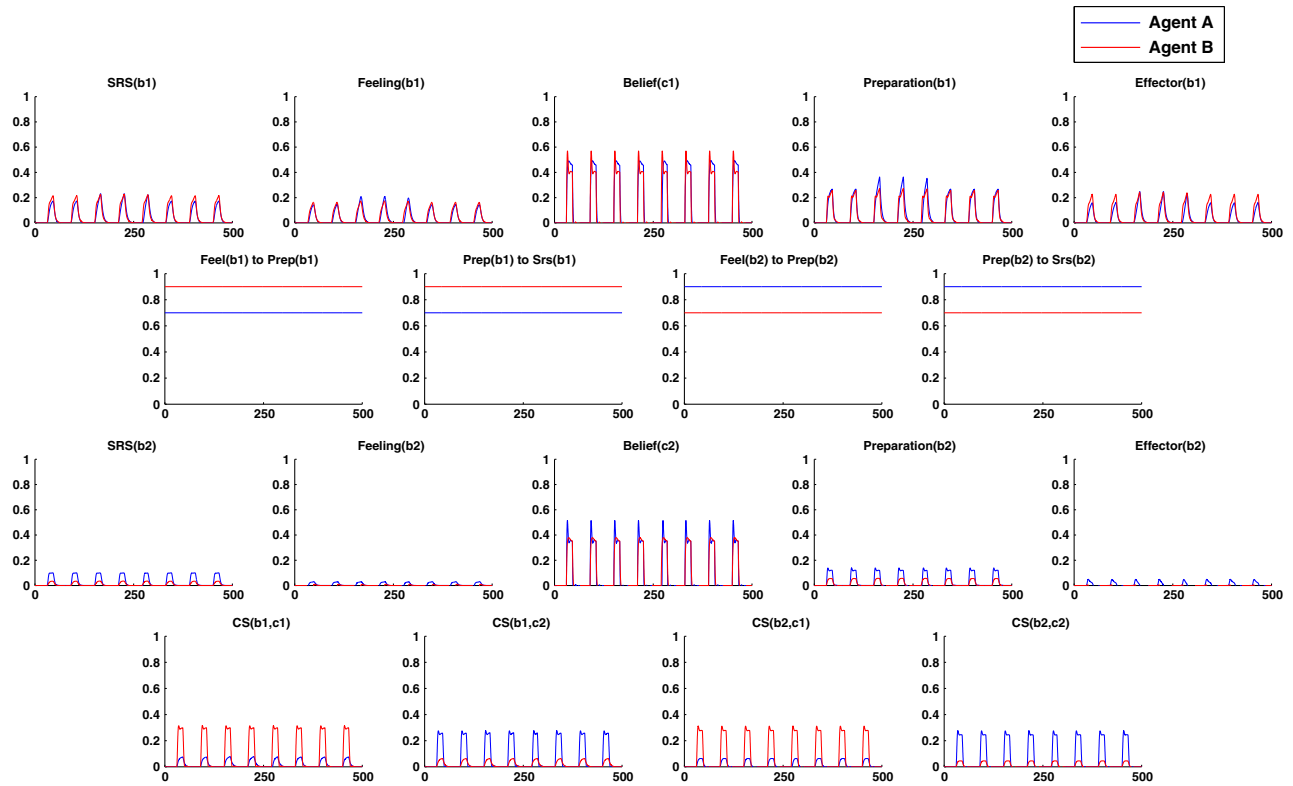


Fig. 5 Effects of contagion on behaviour with regulation in both agents, and without learning. In this case there is no learning of behaviour, therefore the values for the connection strengths (row 2) are constant (as in Fig. 4). The graphs for preparation(b_i) and effector(b_i) show that almost no kind of behaviour occurs because of the strong form of regulation in both agents. The values for agent A's emotion regulation connections are $wgt_bel_cs = [0.1, 0.5; 0.1, 0.5]$, $wgt_cs_bel = [-1, -1; -1, -1]$, $wgt_feel_cs = [0.1, 0.1; 0.1, 0.1]$, and $wgt_cs_feel = [-0.1, -0.1; -0.1, -0.1]$. Similarly for agent B; the only exception is $wgt_bel_cs = [0.5, 0.1; 0.5, 0.1]$.

ciation with option 1. Interaction between the agents depend on the social settings which is captured by: expressiveness ε_A and ε_B of the agents, the channel strengths α_{BA} and α_{AB} from B to A and from A to B respectively, and also on the degree to which an agent is open for influences of others in general (openness δ_A and δ_B). Interaction starts during the time period from 150 to 300 (days: 3, 4 and 5), in which mainly agent B affects agent A positively for option 1. Finally agent A indeed takes over the choice for option 1. But this fully depends on agent B's presence. Since agent A's preparedness for option(b_1) depends on the presence of agent B, as soon as the link between the agents is cut at the end of phase 2 (after time point 300), agent A does not show the preparedness for option 1 anymore. Nothing has been learnt from agent B.

Direct effects of combined contagion and regulation on behaviour, without learning

The second scenario is a different scenario with the same settings but this time with emotion regulation realised; see the settings in Table 3. The other settings are the same. Identical to the case in Section 'Direct effects of contagion without regulation on behaviour, and without learning', the

contagion starts during the same time period but now in the presence of a regulation mechanism. Note that this mechanism works in both agents. Within both agents it makes the activation levels of feelings lower. Regulation within both agents takes place based on the control states $cs(b_i, c_j)$. An appropriate combination $cs(b_i, c_j)$ is used to control the feelings for the specific option. The parameter values to control the feelings are given in Table 3. The results for this case are shown for three distinguished cases in Figs. 5–7 where the contagion is active in all three cases, but the regulation is working either for agent A (Fig. 6), or for agent B (Fig. 7), or for both agents (Fig. 5). In the case shown in Fig. 5, where regulation is working for both agents, all four instances of the control state, i.e., $cs(b_1, c_1)$, $cs(b_1, c_2)$, $cs(b_2, c_1)$, and $cs(b_2, c_2)$ are working to achieve the combined regulation. Fig. 6 illustrates the results for emotion regulation in agent A; the control state combination $cs(b_1, c_2)$ and $cs(b_2, c_2)$ is used here for the emotion regulation. Although emotion regulation has effects on the behaviour (option(b_2)), for the time period in which interaction takes place, agent A receives contagion effects. Because of that the blue spikes can be seen for that time period (phase 2: time points 150–300). In Fig. 7 emotion regulation for agent B is addressed, using the combination $cs(b_1, c_1)$ and $cs(b_2, c_1)$ to perform this emotion regulation. In this case Agent B con-

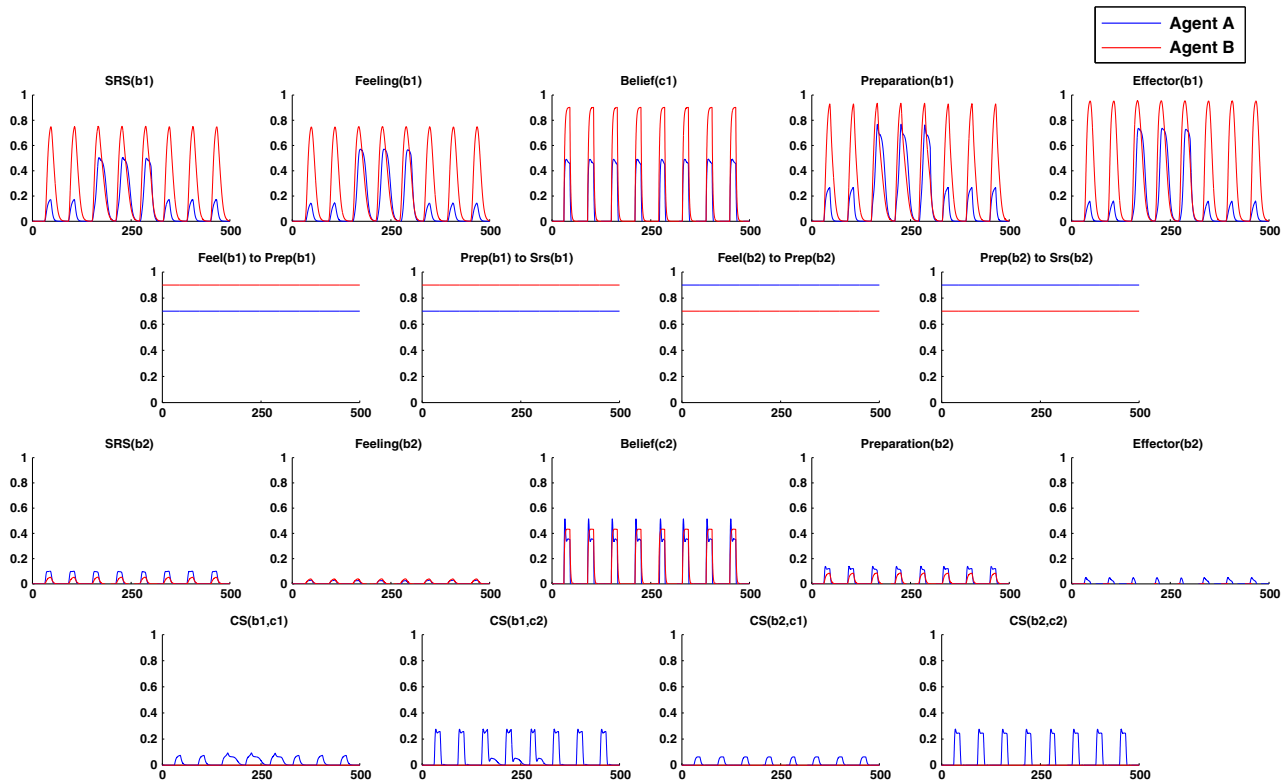


Fig. 6 Effects of contagion on behaviour with regulation only in agent A, and without learning. The scenario shows the contagion effects in agent A during a certain time (see graph for preparation(b_1)) but it is not lasting because the learning of behaviour does not happen. For the learning connections and emotion regulation connections in agent A the values are similar to those in Fig. 5 but for agent B the values are zero for all emotion regulation connections.

trol the feelings and belief for option 1 therefore the contagion has no decisive effect on agent A's behaviour.

Effects of contagion without regulation on behaviour and learning of behaviour

The results for this scenario were already depicted in Fig. 3. In this section the role of contagion on learning of behaviour is discussed in more detail; it can be seen from Fig. 1 that two connections are learnt. The first one is from preparation state $prep(b_i)$ to sensory representation $srs(b_i)$ and the second one is from feeling state $feel(b_i)$ to preparation $prep(b_i)$. The learning is based on the Hebbian learning principle: when both nodes are active simultaneously the connection is strengthened. The learning process starts from connection weights with very low values (initially set at 0.1 for both connections), but as the contagion starts to take place, the learning for agent A gradually increases the connection weights from lower to higher values. It is observed in agent A that, when the contagion process is stopped after time point 300, the spikes for the activation level for $prep(b_1)$ and $eff(b_1)$ remain much higher than they were before time point 150. This shows that due to the social interaction behaviour has been learnt that persists as individual behaviour also without a social context. For

both connections learning rate η is 0.25, and extinction rate ζ is 0.0001. The other parameter values are the same as in the above scenario. In the subsequent section it is shown how a regulation mechanism can disrupt the learning of a behaviour.

Effects of contagion combined with regulation on behaviour and learning of behaviour

The aim of this experiment is to observe the learning in the presence of regulation. As with the previous cases contagion takes place between the time point 150 and 300. All parameter values are identical to previous scenarios. The model has two connections that are learnt, one is from $feel(b_i)$ to $prep(b_i)$ state and the other one is from $prep(b_i)$ to $srs(b_i)$. The learning rate and the extinction rate for both connections are 0.25 and 0.0001, respectively. The learnt connections shape the decision making process in an agent. The learning process in the presence of a regulation mechanism may not be very effective. For example, when agent A is affected by agent B to adapt to good behaviour, agent A may not completely learn this behaviour. This may happen because the emotion regulation weakens the emotion to stimulate the behaviour. Fig. 8 shows a scenario in which regulation is active in agent A. Because of this regulation

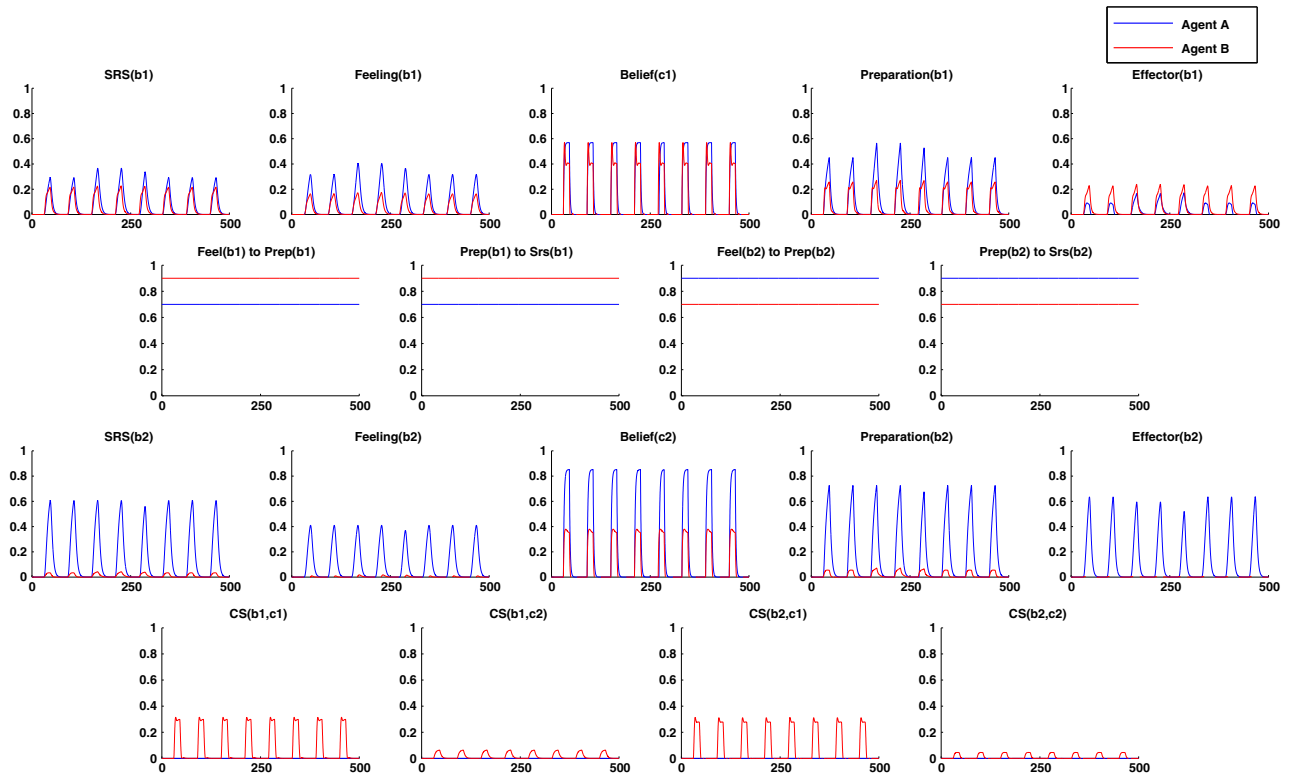


Fig. 7 Effects of contagion on behaviour with regulation only in agent B, and without learning. The connection weights for agent B have the same values as in Fig. 5 except for agent A which are set at 0. It is observed that in this case the contagion does not have effects on agent A.

mechanism the learning of behaviour does not take place in its entirety; this can be seen in the second row of Fig. 8. Besides, it also has a strong effect on option 2 as Fig. 8 illustrates that activation levels (blue spikes) of states $\text{prep}(b_2)$ and $\text{eff}(b_2)$ are low. A slight variation of the previous scenario is presented in Fig. 9 by lowering the channel strength from agent B to A (to 0.4). The purpose of this case is to demonstrate that lower connection strength makes less contagion and that makes less learning. A scenario in which regulation takes place for agent B is depicted in Fig. 10. Since the contagion is very strong from agent B to agent A, some learning takes place for the good option, i.e., b_1 , but not much. This happens because the learning process itself for agent B is disturbed by the regulation process. Finally, Fig. 11 illustrates a case in which regulation is active for both agents which results in no behaviour (neither positive nor negative) and the learning process also does not occur.

Discussion

In this paper the integration of emotion regulation and emotion contagion in socially affected decision making processes was addressed. As a point of departure, decision making is assumed to be based on valuing of predictions involving feeling states generated in the amygdala (Damasio, 1994; Ochsner & Gross, 2014; Phelps et al.,

2014; Rolls, 2013, 2014). The presented model is adaptive based on Hebbian Learning (Hebb, 2002). The model can prove to be useful in circumstances where it is important to get rid of bad habits and adapt a healthy lifestyle. Analysis of the model is done using different scenario settings. For instance it is observed in a simulation trace that the emotion regulation mechanism does not only control the feelings and beliefs of agents in a non-learning context, but it is also effective in an environment where learning of behaviour occurs over a certain period of time.

The main novelty of the presented model is in the integration of different processes addressed in separate existing models. It is quite common to focus computational models on one particular subprocess, thereby neglecting other processes that take place at the same time. This deviates from real human processes in which it is not often possible to put one process on hold in order to focus on another process. In reality most often different processes work at the same time and affect each other by their interaction. The way in which computational models often only address single processes in isolation leads to a blind spot for such interactions. Therefore it is an important challenge to work more on integrative models in which different processes are modelled, including their interaction. For example, models have been introduced for social contagion (Bosse, Duell, Memon, Treur, & Van Der Wal, 2009; Bosse et al., 2014) for emotion regulation (Bosse, Pontier, & Treur, 2010; Gross, 2015a, 2015b) and for decision making (Treur & Umair, 2011) sepa-

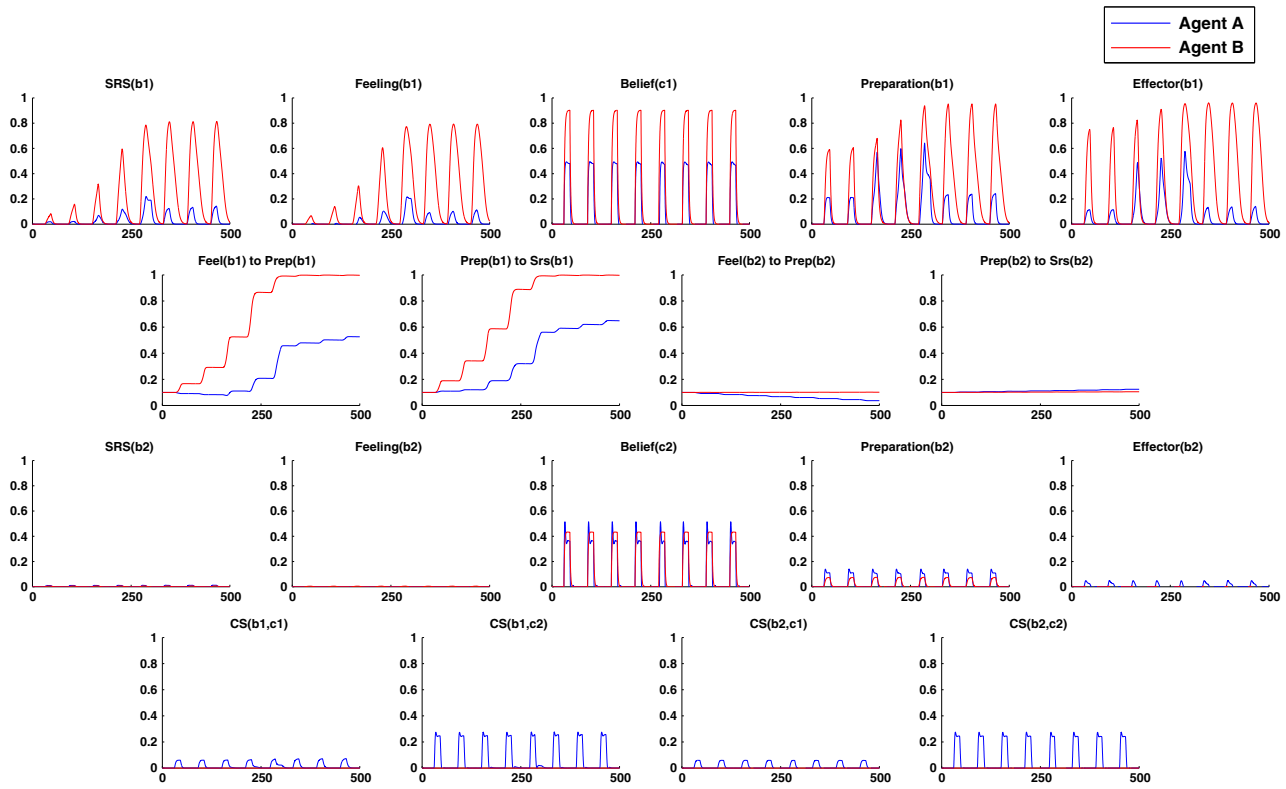


Fig. 8 Effects of contagion and learning in agent A in the presence of a regulation mechanism. The emphasis is on the learning connections (row 2) for option b_1 . The learning for the agent A occurs but not completely because of emotion regulation; e.g., the graphs $\text{feel}(b_i)$ to $\text{prep}(b_i)$ and $\text{prep}(b_i)$ to $\text{srs}(b_i)$ help understand the scenario. The initial values for both are 0.1. To enable emotion regulation in agent A the following weights were used $\text{wgt_bel_cs} = [0.1, 0.5; 0.1, 0.5]$, $\text{wgt_cs_bel}_i = [-1, -1; -1, -1]$, $\text{wgt_feel_cs} = [0.1, 0.1; 0.1, 0.1]$, and $\text{wgt_cs_feel}_i = [-0.1, -0.1; -0.1, -0.1]$.

ately, but the integration of these different processes was never addressed; therefore there is no computational coverage of their interaction. For these reasons, an integration of these processes was taken as the focus and novelty of the current paper.

In this paper a general approach was presented; no specific types of feelings have been considered. In future work it may be interesting to differentiate into specific kinds of feelings, and make emotion regulation specific to a particular kind of emotion. Another future direction would be to consider a reward mechanism after an action has been performed, as decisions related to a particular choice are often also based on a prediction of a rewarding or aversive consequence experienced in the past. It would also be interesting to study the role of emotion regulation within such a reward mechanism. The current model may be used as a point of departure for this.

The scenario and model presented in this paper can be used as a basis for an application providing personalised support taking into account an individual's affective states. This can be done in the context of ambient intelligence and affective computing. For example, such an application can suggest appropriate interventions required to help people in a social network to improve their physical activity level. This may be done by identifying individuals in a social

network (Klein, Manzoor, Mollee, & Treur, 2014) that could motivate and support a person (who is not so motivated) to join a sport school or exercise regularly in order to improve physical health. An intelligent coaching system can be designed which can have different components based on modern technologies, for instance a smart phone or tablet that can be used to send persuasive messages regarding different coaching tips. In addition the more advanced smart phones are often equipped with various kinds of digital sensors that help to determine the context of an individual and achieve personalisation by continuous monitoring. Moreover, social sensors can form a basis for achieving dedicated contextual and social support and social influence. A reasoning system based on this kind of model can be built which acts as the engine for such kind of system. The prime objective of such a (socially aware) system is to understand and reason about the human mental states, detect the causes of unhealthy behaviour and provide tailored information and motivational messages to help individuals adapt a more healthy lifestyle.

Finally, the model presented in this paper can also be a basis for more contextually and socially tuned emotions shown by virtual agents or humanoid robots, as far as it involves a social context in which contagion takes place.

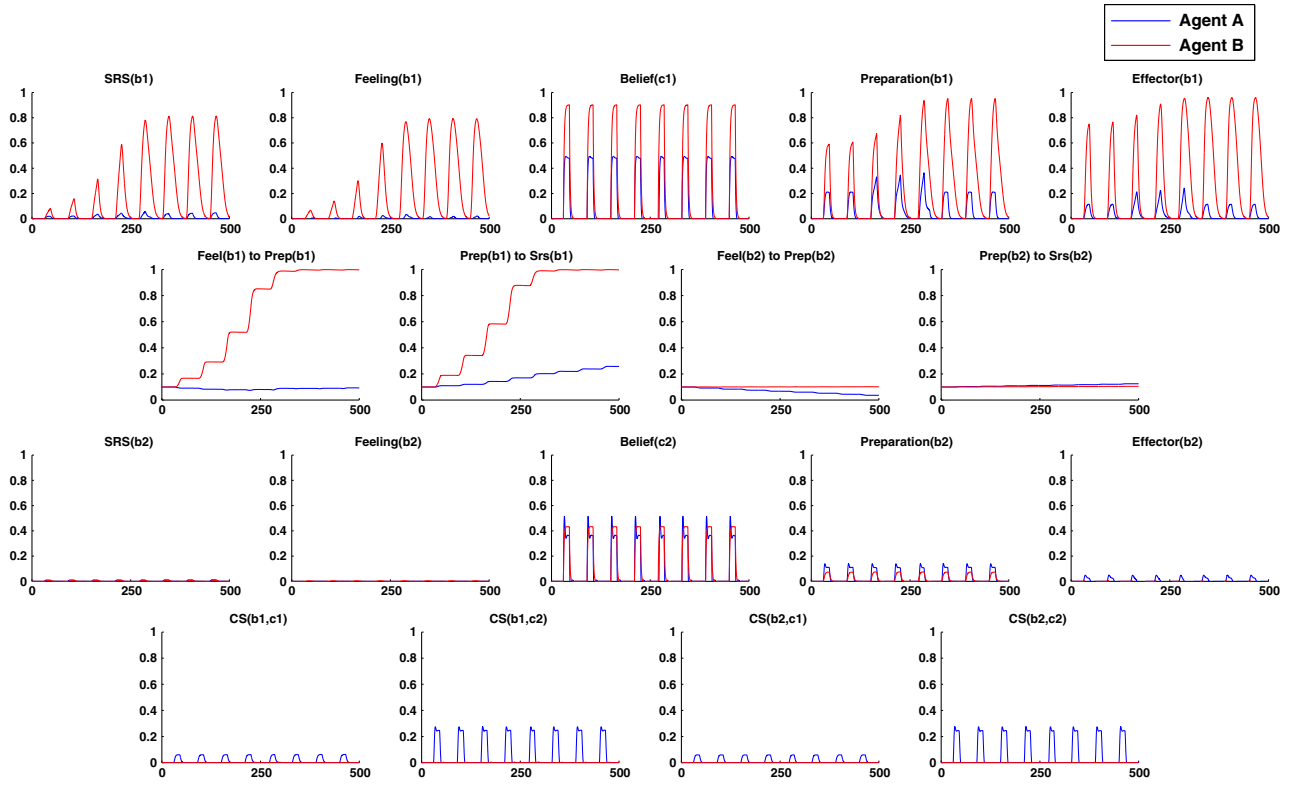


Fig. 9 Effects of contagion with lower channel strength and learning in agent A in the presence of a regulation mechanism. Again the focus is on the learning (row 2 connections $\text{feel}(b_1)$ to $\text{prep}(b_1)$ and $\text{prep}(b_1)$ to $\text{srs}(b_1)$) of agent A. Similar values were used except for channel strength = 0.4.

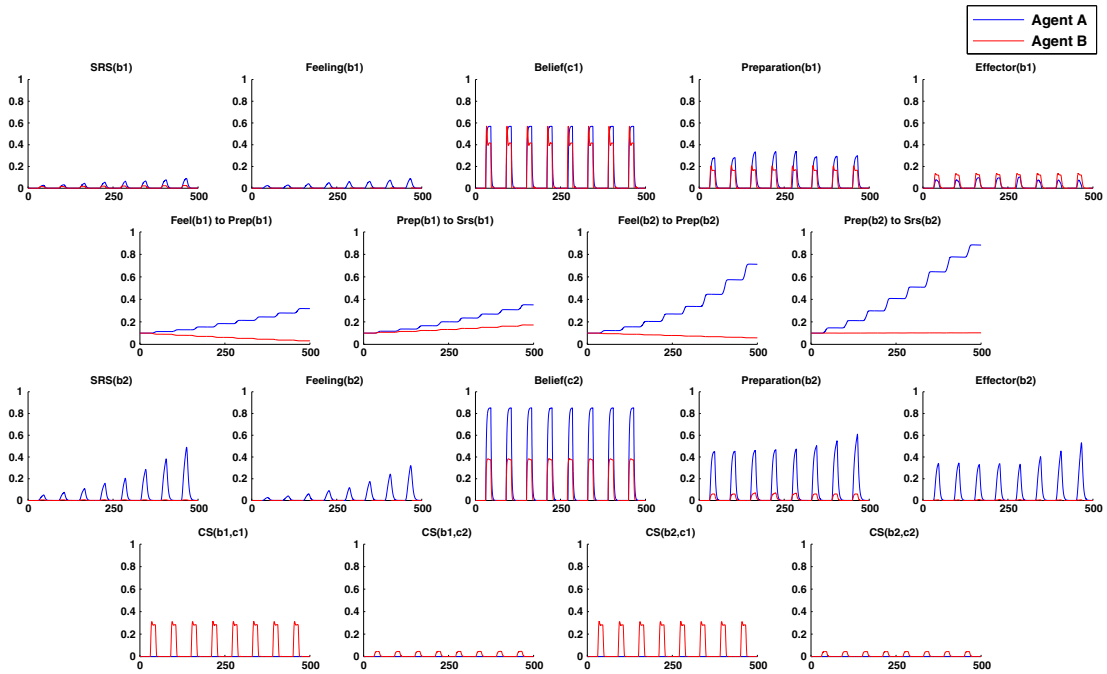


Fig. 10 Regulation on learning of behaviour in agent B. The weight values $\text{wgt_beli_cs} = [0.5, 0.1; 0.5, 0.1]$, $\text{wgt_cs_beli} = [-1, -1; -1, -1]$, $\text{wgt_feeli_cs} = [0.1, 0.1; 0.1, 0.1]$, and $\text{wgt_cs_feeli} = [-0.1, -0.1; -0.1, -0.1]$. Connections $\text{feel}(b_i)$ to $\text{prep}(b_i)$ and $\text{prep}(b_i)$ to $\text{srs}(b_i)$ starts at 0.1.

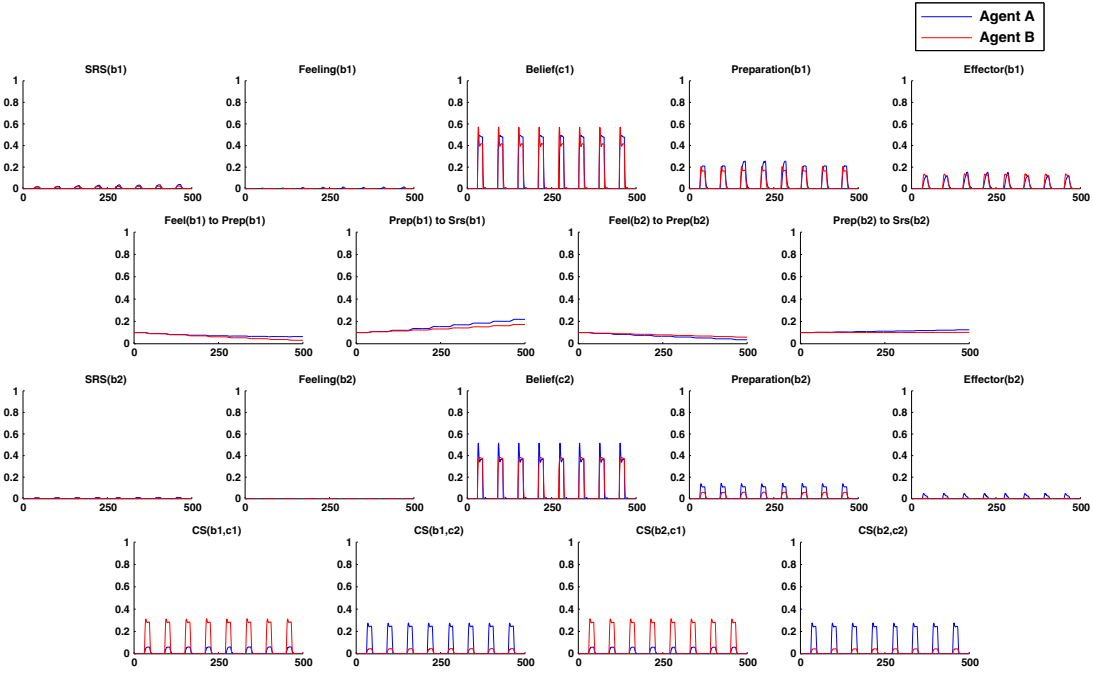


Fig. 11 Regulation and learning of behaviour in both agents. The values for agent A's emotion regulation connections were $wgt_bel_cs = [0.1, 0.5; 0.1, 0.5]$, $wgt_cs_bel_i = [-1, -1; -1, -1]$, $wgt_feel_cs = [0.1, 0.1; 0.1, 0.1]$, and $wgt_cs_feel_i = [-0.1, -0.1; -0.1, -0.1]$. Similar values for agent B with only one exception which is $wgt_bel_cs = [0.5, 0.1; 0.5, 0.1]$. Connections $feel(b_i)$ to $prep(b_i)$ and $prep(b_i)$ to $srs(b_i)$ starts at 0.1.

Table 5 Formal specification of the model in LEADSTO format.

LP1	$EA(a, V_1) \ \& \ WS(b, V_2) \rightarrow WS(b, V_2 + \gamma [f(w_2V_1) - V_2] \ \Delta t)$
LP2	$WS(s, V_1) \ \& \ SS(s, V_2) \rightarrow SS(s, V_2 + \gamma [f(w_4V_1) - V_2] \ \Delta t)$
LP3	$SS(s, V_1) \ \& \ SR(s, V_2) \rightarrow SR(s, V_2 + \gamma [f(w_7V_1) - V_2] \ \Delta t)$
LP4	$SR(b_{obj}, V_1) \ \& \ PA(a_{obj}, V_2) \ \& \ PO(a, b, c, s, V_3) \ \& \ SR(b_{obj}, V_4) \rightarrow SR(b_{obj}, V_4 + \gamma [f(w_9V_1, w_{10}V_2, w_{11}V_3) - V_4] \ \Delta t)$
LP5	$PA(a_{sub}, V_1) \ \& \ SR(b_{sub}, V_2) \rightarrow SR(b_{sub}, V_2 + \gamma [f(w_{12}V_1) - V_2] \ \Delta t)$
LP6	$SR(c, V_1) \ \& \ SR(s, V_2) \ \& \ PD(b, V_3) \ \& \ F(b_{obj}, V_4) \ \& \ PA(a_{obj}, V_5) \rightarrow PA(a_{obj}, V_5 + \gamma [f(w_{13}V_1, w_{14}V_2, w_{15}V_3, w_{16}V_4) - V_5] \ \Delta t)$
LP7	$F(b_{sub}, V_1) \ \& \ CD(b, V_2) \ \& \ SR(s, V_3) \ \& \ F(b_{obj}, V_4) \ \& \ RAWr(a, b, c, s, V_5) \ \& \ PAwr(a, b, c, s, V_6) \ \& \ PA(a_{sub}, V_7) \rightarrow$ $PA(a_{sub}, V_7 + \gamma [f(w_{17}V_1, w_{18}V_2, w_{19}V_3, w_{20}V_4, w_{21}V_5, w_{22}V_6) - V_7] \ \Delta t)$
LP8	$SR(c, V_1) \ \& \ SR(s, V_2) \ \& \ SR(b_{obj}, V_3) \ \& \ PD(b, V_4) \rightarrow PD(b, V_4 + \gamma [f(w_{23}V_1, w_{24}V_2, w_{25}V_3) - V_4] \ \Delta t)$
LP9	$SR(s, V_1) \ \& \ SR(b_{obj}, V_2) \ \& \ CD(b, V_3) \rightarrow CD(b, V_3 + \gamma [f(w_{26}V_1, w_{27}V_2) - V_3] \ \Delta t)$
LP10	$SR(b_{obj}, V_1) \ \& \ PD(b, V_2) \ \& \ F(b_{obj}, V_3) \rightarrow F(b_{obj}, V_3 + \gamma [f(w_{28}V_1, w_{29}V_2) - V_3] \ \Delta t)$
LP11	$SR(b_{sub}, V_1) \ \& \ CD(b, V_2) \ \& \ F(b_{sub}, V_3) \rightarrow F(b_{sub}, V_3 + \gamma [f(w_{30}V_1, w_{31}V_2) - V_3] \ \Delta t)$
LP12	$SR(c, V_1) \ \& \ F(b_{obj}, V_2) \ \& \ PA(a_{obj}, V_3) \ \& \ RO(a, b, c, s, V_4) \ \& \ PO(a, b, c, s, V_5) \rightarrow PO(a, b, c, s, V_5 + \gamma$ $[f(w_{32}V_1, w_{33}V_2, w_{34}V_3, w_{35}V_4) - V_5] \ \Delta t)$
LP13	$F(b_{sub}, V_1) \ \& \ PA(a_{sub}, V_2) \ \& \ PO(a, b, c, s, V_3) \ \& \ F(b_{obj}, V_4) \ \& \ RAWr(a, b, c, s, V_5) \ \& \ PAwr(a, b, c, s, V_6) \rightarrow$ $PAwr(a, b, c, s, V_6 + \gamma [f(w_{36}V_1, w_{37}V_2, w_{38}V_3, w_{39}V_4, w_{40}V_5) - V_6] \ \Delta t)$
LP14	$PAwr(a, b, c, s, V_1) \ \& \ PA(a_{sub}, V_2) \ \& \ PO(a, b, c, s, V_3) \ \& \ PA(a_{obj}, V_4) \ \& \ EA(a, V_5) \rightarrow EA(a, V_5 + \gamma$ $[f(w_{41}V_1, w_{42}V_2, w_{43}V_3, w_{44}V_4) - V_5] \ \Delta t)$
LP15	$PA(a_{sub}, V_1) \ \& \ SR(c, V_2) \ \& \ PO(a, b, c, s, V_3) \ \& \ F(b_{obj}, V_4) \ \& \ EA(a, V_5) \ \& \ RO(a, b, c, s, V_6) \rightarrow RO(a, b, c, s, V_6 + \gamma$ $[f(w_{45}V_1, w_{46}V_2, w_{47}V_3, w_{48}V_4, w_{49}V_5) - V_6] \ \Delta t)$
LP16	$F(b_{sub}, V_1) \ \& \ PAwr(a, b, c, s, V_2) \ \& \ RO(a, b, c, s, V_3) \ \& \ F(b_{obj}, V_4) \ \& \ RAWr(a, b, c, s, V_5) \rightarrow RAWr(a, b, c, s, V_5 + \gamma$ $[f(w_{50}V_1, w_{51}V_2, w_{52}V_3, w_{53}V_4) - V_5] \ \Delta t)$
LP17	$RAWr(a, b, c, s, V_1) \ \& \ RO(a, b, c, s, V_2) \ \& \ EO(a, b, c, s, V_3) \rightarrow EO(a, b, c, s, V_3 + \gamma [f(w_{54}V_1, w_{55}V_2) - V_3] \ \Delta t)$

Appendix A

Formal specification in the hybrid LEADSTO format (Bosse et al., 2007) is shown in Table 5. LEADSTO is a hybrid modelling language in which a dynamic property or temporal

causal relation $a \rightarrow b$ denotes that when a state property a (or conjunction thereof) occurs, then after a certain time delay, state property b will occur. The time delay defined in LEADSTO is taken as a uniform time step Δt here.

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